ment or wherever, and if it can gain public support as well, there will then have been created a solid base upon which to build a technology of physician power. There is considerable evidence that knowledge is power, involvement is power, money is power (and, by the way, it is possible to use other people's money to exercise power) and public support is power. Given a common purpose, the technology for use of these forms of power readily can be developed and applied in the scientific, social, economic and political arenas of health care.

Third, it is quite apparent that physician power can be exercised most effectively through teamwork. While physicians may be the leading professionals when it comes to knowledge of health and its disorders, they share with others their recognition of individuality in human nature, and share with many others their commitment to the betterment of human health and well-being. Teamwork is certainly essential for the efficient and effective exercise of physician knowledge and physician power. There is and must be teamwork in patient care, teamwork in health care, teamwork in public education, teamwork in achieving high quality care and cost benefits for physicians and patients alike, and teamwork in achieving political and legislative goals. There must be teamwork within the profession and with others, including government, if physician power is to find full expression.

Physician power of this kind can become a potent force for the betterment of health care in this nation and, if properly developed and exercised, it can achieve results which will go far to reduce insecurity, frustration and resentment among practicing physicians in the care of their patients. But there must be an identifiable purpose that is acceptable not only to the vast majority of physicians but to the public, there must be better development of a more sophisticated technology for the use of physician power, and there must be an even greater recognition of the importance and implications of teamwork in support of the common purpose.

—MSMW

Allergic Rhinitis

ALLERGIC RHINITIS AFFECTS one and one-half million Californians and over 13.5 million Americans.[1] Further, there is a similar number of persons receiving anti-histaminic drugs for mild but annoying nasal symptoms, many of which are related to allergic sensitivity. Such people are not working at their peak efficiency due to impaired sleep and drug side effects. A complication of allergic rhinitis is serous otitis which affects two and a half million American children and causes partial deafness and difficulty in learning.[2] Allergic rhinitis, then, is a leading health problem in numbers of Americans affected, and the costs in outlays for drugs, in reduced working efficiency and in personal comfort are huge.

In the conference reported elsewhere in this issue by Mendelson et al, the mechanism of immunoglobulin E-mediated allergic reactions is reviewed, especially with regard to classical allergens such as pollen, house dust and animal danders. Further, they point out the "priming effect" of a heavy exposure to ragweed pollen antigen which causes fragmentation of the nasal basement membrane which makes the nose vulnerable to much smaller exposures of pollen and other allergens.[3] This may occur with other allergens also and may account for the common cyclic occurrence of allergic symptoms, especially in California, where seasonal swings in pollen counts are less pronounced but pollens occur the year around. The authors also refer to milk and other foods which may cause allergic respiratory symptoms, often also on a cyclic basis. Further, they mention elevated serum IGE in a patient with hayfever. Several methods utilizing radiolabeled antigens or anti-IGE sera are proving useful in quantitating specific IGE antibodies in allergic patients.[4,5] These are useful in following a patient's course during treatment.

It must be emphasized, however, that not all rhinitis is allergic, as is illustrated by vasomotor rhinitis, for which the precise pathogenic mechanism is unknown. Often such cases are labeled "allergic" for want of a diagnosis, and temporizing anti-allergic therapy is given with negative results. This calls into question all such therapy in general. Connell[6] described "nasal mastocytosis" in non-allergic patients who had abundant histamine release and rhinitis. Other investiga-

tors[7] are describing specific nasal pathophysiology which removes other cases from the diagnostic discard bag of vasomotor rhinitis.

Under specific therapy for allergic diseases, avoidance of the antigen, where possible, is still the cardinal principle. The UC, San Diego, group have begun a unique and important program of training paramedical specialists in environmental control; such persons can point out to the patient in his own home specific sources of possible allergens and how to avoid them. Such environmental technologists should be a great asset to allergists and other physicians in private or clinic practices.

Immunotherapy for allergic diseases remains under attack for efficacy despite its 60-year history.[8] However, in ragweed hayfever, "the purest form of allergy," a number of double-blind studies have shown unequivocal improvement at statistically significant levels[9,10] in treated patients compared with placebo-treated controls.

Non-specific therapy includes classical use of nasal decongestants and anti-histaminic drugs which compete with histamine for receptors in nasal vessels. With the demonstration that β-adrenergic agonists raise cellular levels of cyclic 3'5' adenosine mono-phosphate (AMP) which inhibits histamine release from sensitized human leukocytes, there is renewed interest in these agents in all allergic diseases, including allergic rhinitis.[11] Disodium cromoglycate inhibits histamine and SRS-A release from human lung mast cells in asthma and appears to be useful therapeutically.[12] Therefore, its use in allergic rhinitis is also being explored, so far with variable but some promising results.[13,14]

Over the past decade, great strides have been made in defining the immunologic and pharmacologic mechanisms of allergic reactions which are being now translated into new and effective treatments.

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The Treatment of Acute Myelocytic Leukemia

IN 1961 COMPLETE remission (CR) was achieved in only 5 percent of patients treated for acute myelocytic leukemia (AML).[1] Since the development and use of cytosine arabinoside and daunomycin, increasing remission rates have been reported.[2,3] The paper of Poth et al in this issue of California Medicine also reports higher remission rates but stimulates many questions concerning the treatment of AML. While the authors address themselves to the achievement of remission, the reader should bear in mind that the ultimate goal of treatment is not only remission but survival of good quality.

In 1966, Dameshek et al[4] reported that patients with "smoldering" acute leukemia lived longer than those who achieved complete remission, and concluded that there was no constant relation between achievement of remission and length of survival. With newer treatment programs, Dameshek's conclusions of six years ago may be invalid today, but it is still necessary to evaluate the quality and length of survival beyond the achievement of remission before the effectiveness of therapy can be judged. A program in which all patients achieve remission but die the next day is obviously inferior to one in which 50 percent of patients who achieve remission go on to a prolonged survival. One can also ask how well the remission-inducing regimen prepares a patient for maintenance therapy and survival.